

Effluent Characteristics and Concerns

5.1 Effluent Overview

Treated effluent from a wastewater treatment plant may contain minimal quantities of many different substances that could impact physical, biological, and/or human environments when it is discharged. Of primary concern are:

- Nutrients
- Bacteria
- Metals, Volatiles, Semi-Volatiles, Phenolics, and Polychlorinated Biphenyls (PCBs)
- Pharmaceuticals and Personal Care Products (PPCPs)
- Salinity

As stated previously, the RBWWTP is an advanced secondary treatment plant that produces an effluent of higher quality than that of a typical secondary treatment plant. However, it is the intent of this risk assessment to consider not only normal operation but also worst-case scenarios. For that purpose, the level of quality of wastewater at the lower levels of treatment described in Table 5-1 must also be investigated.

Table 5-1 Description of different levels of treatment at a WWTP

Type Wastewater	Disinfected?	Description
Raw	No	Untreated wastewater as it enters a wastewater treatment plant
Primary Effluent	No	Wastewater that has undergone preliminary treatment including screening and grit removal followed by settling to remove the coarser and solids and heavier particulate matter
Secondary Effluent	No	Wastewater has undergone preliminary treatment (grit and screening), primary settling, secondary treatment consisting of a biological process for the removal of organics and nitrogen, chemical addition for phosphorus removal and secondary settling
Tertiary Effluent	Yes	Secondary effluent which has undergone additional treatment including filtration and disinfection using chlorine.

5.2 Nutrients

Nutrients are "any element or compound essential as a raw material for organism growth and development, including but not limited to nitrogen and phosphorus" (DNREC 2004). As mentioned in Chapter 3, alternative methods of effluent discharge were evaluated and considered due to high levels of nitrogen and phosphorus in Rehoboth Bay (DNREC 1998). The impact of these two nutrients on the environment must be evaluated



for each alternative. When discharging to surface water, the effluent must either be discharged at or rapidly diluted to a concentration at which nitrification is no longer expected to occur.

Delaware surface water standards require nitrogen and phosphorus concentrations within Rehoboth Bay to be below submerged aquatic vegetation growth season average levels (DNREC 2004). It is assumed that once nitrogen and phosphorus levels within the effluent dilute to this level, the impact on the environment is minimal. No regulations or standards exist for nitrogen and phosphorus concentrations within the ocean; however, background nutrient concentrations should be met after dilution. Table 5-2 presents the post-dilution nutrient concentration goals within Rehoboth Bay and the Atlantic Ocean. The assumed nutrient concentrations at different levels of treatment are presented in Table 5-3.

Table 5-2 Nutrient Concentration Goals (DNREC 2004)

Standard	Nitrogen Concentration (mg/L as N)	Phosphorus Concentration (mg/L as P)
Surface water standards for Rehoboth Bay	0.14	0.01
Background levels within the ocean	0.37	0.06

Table 5-3 Nutrient concentrations by level of treatment provided

Treatment	Nitrogen Concentration (mg/L as N)	Phosphorus Concentration (mg/L as P)
Raw	30 - 40	6 - 8
Primary Effluent	25 - 30	4 - 6
Secondary Effluent ¹	18 - 25	1 - 2
Tertiary Effluent ²	6 - 10	< 0.5

Notes:

- 1. Conventional activated sludge with nitrification.
- 2. Typical BNR plant with chemical phosphorus removal.



The RBWWTP was upgraded in 1994 and 1997 to incorporate a biological nitrogen removal process and a chemical phosphorus removal process. This was done in order to achieve compliance with the nutrient standards outlined in the "Comprehensive Conservation and Management Plan (CCMP) for Delaware's Inland Bays" (DNREC 1995). These standards were developed for discharge into the Inland Bays and will no longer apply when an alternate form of discharge is in place. However, the City intends to continue to operate the RBWWTP in a manner which still removes nitrogen and phosphorus consistent with the nutrient standards of the 1995 CCMP. The average concentration of Total Nitrogen and Total Phosphorus (January 2007 – July 2010) was 6.2 mg/L TN and 0.35 mg/L TP. To achieve surface water standards for nitrogen and phosphorus within Rehoboth Bay, the effluent must undergo at least a 1:45 dilution. To reach background nutrient levels within the ocean, the effluent must undergo at least a 1:17 dilution.

5.3 Pathogenic Organisms

5.3.1 Indicator Organisms

Raw wastewater contains a wide variety of microbial pathogens (potential disease causing agents) including bacteria, protozoa and viruses. Various types of viruses are routinely found in untreated wastewater including human enteric viruses such as enteroviruses, hepatitis A virus and rotavirus, but they are typically not monitored in the effluent of a wastewater treatment plant because of the difficulty and expense of isolating and analyzing them. The protozoa of concern are primarily Cryptosporidium and Giardia, which have been responsible for waterborne outbreaks of disease. These organisms are removed through a filtration process. Data regarding the removal of bacteria, viruses and protozoa are quite variable, but generally wastewater treatment processes are capable of 2 to 3 log removal of the microbial indicators prior to disinfection (Wen, et al. 2009). Bacterial contamination is monitored by analyzing for a single indicator organism that is representative of the presence of fecal contamination. The indicator organism that is established as the measure of bacterial water quality in Delaware marine surface waters is the Enterococcus. This is in conformance with the EPA requirements established by the BEACH Act in 2001. Enterococcus is a human specific member of the fecal streptococcus group, which has been demonstrated to exhibit high survival rates in saltwater.

5.3.2 Water Quality Criteria

Bacterial water quality standards are provided for primary and secondary contact recreation in both marine and fresh waters by DNREC in Section 4.6.3.3.2 of the Surface Water Quality Standards (DNREC 2004). These standards are in compliance with federal requirements as stated in the Clean Water Act, which was amended in 2001 to include the BEACH Act. The BEACH Act required states with coastal recreational waters to adopt the new water quality standards that were developed by EPA as protective of public health. The criteria for primary contact in marine waters, which has the most stringent standard, is presented in Table 5-4.



Table 5-4 Criteria for primary contact in marine waters (DNREC 2004)

	Maximum allowable enterococcus colonies
Single value	104 Colony Forming Units (CFU) / 100 mL
Geometric mean	35 Colony Forming Units (CFU) / 100 mL

5.3.3 Removal in a Wastewater Treatment Plant

Various studies of the removal of microorganisms by conventional secondary treatment, consisting of aerobic biological treatment followed by gravity settling, indicates that there is significant reduction in the bacterial and viral indicator organisms. Tertiary treatment consisting of effluent filtration (the RBWWTP has effluent filtration) removes additional microorganisms. A study by the National Research Council indicated that secondary treatment removes up to 99% of fecal coliforms and enteric viruses (National Research Council 1998). Chlorination following secondary treatment will further reduce levels of harmful bacteria by greater than 99% or even to non-detect levels (USEPA 1998). A study of pathogen removal in four individual WWTPs providing tertiary treatment (with chlorination) indicated that the overall removal efficiency of total and fecal coliform varied from 4.3 to 5.7 logs (Zhang and Farahbakhsh 2007). Viruses and oocysts tend to be more resistant to inactivation by chlorine disinfection but are also reduced by secondary treatment and chlorination. Viruses are removed in secondary treatment by adsorption onto biological flocs and through predation by other organisms. Tertiary treatment with chlorination will further reduce viruses to some extent. The previously referenced study (Zhang and Farahbakhsh 2007) found that tertiary treatment removed from 0.3 to 2.8 logs of F-specific coliphages and from 0.4 to 2.6 logs of somatic coliphages. Coliphages are viruses that infect coliform bacteria and are therefore useful as indicators of viral contamination due to humans and warm blooded animals. Another study indicated that, in general, tertiary treatment is capable of removing from 99.983 to 99.9999998 percent of enteric viruses (World Health Organization (WHO) 1999).

5.3.4 Concentration in Rehoboth Beach Wastewater

The RBWWTP provides a tertiary level of treatment and the effluent is disinfected by chlorination. The WWTP is required by its NPDES permit to reduce the level of enterococcus to less than 14 colonies per 100 mL. This permit level was established based on the fact that the plant currently discharges to the Inland Bays with the potential to impact shellfish beds. The discharge limit is more stringent than the standard required for primary contact in marine waters. The RBWWTP typically performs at a higher level than the required standard, reducing enterococcus levels to well below the maximum allowable value. Effluent enterococcus levels measured at the plant from 2007 and 2009, was often zero with a geometric mean of 2, although one sample was as high as 40 colonies per 100 mL.

Wastewater treatment facilities do not routinely test for the various types of pathogenic organisms in the raw wastewater or through the various stages of a treatment process. Only the testing required for compliance monitoring (enterococcus) is performed. However, to provide a perspective on the typical concentrations present, Table 5-5 presents the level of indicator organisms typically present in wastewater at the levels of treatment described in Table 5-1. This information is based on studies presented in the literature conducted at other wastewater treatment facilities and is representative of typical domestic wastewater.



Table 5-5 Pathenogenic and indicator organisms typically present by level of treatment provided (No. per 100 mL)

Type Wastewater	Oocysts	Total Coliform	Fecal Coliform	Entero- coccus	Virus	Ref	Note
Raw					9.0 x 10 ³	1	Α
	1.55 x 10 ²		3.4×10^6	2.4 x 10 ⁵	1.54×10^2	3	1
	1.25 x 10 ³		11 x 10 ⁶	1.0 x 10 ⁶	1.25 x 10 ³	3	J
	4.9 x 10 ⁴	2.4 x 10 ⁷	9.0 x 10 ⁵	5.0 x 10 ⁵	3.8 x 10 ⁵	4	C, I
		6.3 x 10 ⁷	3.2 x 10 ⁶	1.0 x 10 ⁶	2.5 x 10 ³	5	D, I
			50 x 10 ⁶			6	
	10 ⁶ max					3	
		2.5 x 10 ⁷			2.9 x 10 ⁵	9	F
					6.1 x 10 ⁴	10	G
				5.2 x 10 ⁵		12	
	0.72 x 10 ³ to 1.46 x 10 ³				10 ³ to 10 ⁴	13	
Primary			2.5 x 10 ⁶	1.55 x 10 ⁵	1.45 x 10 ³	3	I
Effluent			9.2 x 10 ⁶	3.0 x 10 ⁵	1.25 x 10 ⁴	3	J
		5.0 x 10 ⁷	3.0 x 10 ⁷	3.6 x 10 ⁶		2	В
				1.0 x 10 ³	3.0 x 10 ²	7	Е
				3.8 x 10 ⁵		12	
					17 to 5,000	13	
Secondary					100 to 1,700	1	А
Effluent (without chlorination)		1.7 x 10 ⁵	7.8 x 10 ³	2.2 x 10 ³	1.8 x 10 ³	4	
		4 x 10 ⁴ to 2.5 x 10 ⁵				8	
			10 ⁴		5 x 10 ²	9	
Secondary					1	1	Α



Type Wastewater	Oocysts	Total Coliform	Fecal Coliform	Entero- coccus	Virus	Ref	Note
Effluent (with chlorination)				4.4		12	
					<1 - 4,700	13	
Tertiary		0.58	0.5	0.45	0	4	
Effluent (with chlorination)		1.6	<1	<1	<1	5	
			10 ²		10	9	
		3	0			11	Н
				5.6		12	
	<1 to 30				<1 to 1.7	13	

References

- 1. (Mukhtasor, Lye and Sharp 2002)
- 2. Orange County Sanitation District study
- 3. (Payment, Plante and Cegka 2001)
- 4. (Rose, et al. 2001)
- 5. (Harwood, et al. 2005)
- 6. URS Study
- 7. (Tree, Adams and Lees 2003)
- 8. (Paraskeva and Graham 2005)
- 9. (Zhang and Farahbakhsh 2007)
- 10. (Myrmel, et al. 2006)
- 11. (Nelson, et al. 2003)
- 12. (Srinivasan, et al. 2011)
- 13. (World Health Organization (WHO) 1999)
- 14. (Wen, et al. 2009)

Notes:

- A. Based on rotavirus
- B. Blend of secondary and primary effluent no disinfection
- C. Oocysts based on giardia virus
- D. Based on enterovirus and giardia cysts



- E. Based on enterovirus
- F. Based on somatic colaphages approximate mean values taken from graphs of log count
- G. Based on F-phages virus mean values
- H. Effluent values based on 7 day medians
- I. Mean value
- J. Max Value

Treated wastewater also poses a risk of contaminating receiving waters with pathogenic bacteria and viruses. The list of pathogens and the resulting disease are summarized in Table 5-6 (Majeti and Clark 1980). Vigneswaran and Sundaravadivel (2004) summarized the normal survival periods of different pathogenic organisms under various conditions (see Table 5-7).

Table 5-6 Pathogenic organism and resulting disease (Majeti and Clark 1980)

Type of pathogen	Disease
Viruses:	
Hepatitis virus A	Hepatitis A
Bacteria:	
Escherichia coli	Gastroenteritis (symptoms include diarrhea, nausea, dehydration, and lack of febrile response)
Salmonella spp.	Salmonellosis (symptoms include diarrhea, abdominal cramp, fever, nausea and vomiting)
Shigella spp.	Shigellosis a.k.a. bacillary dysentery (symptoms include acute diarrhea)
Vibrio cholerae	Cholera (symptoms include acute diarrhea, vomiting, dehydration, lowered body temperature and blood pressure)
Protozoa:	
Entamoeba hystolyticalysts	Amoebiasis a.k.a Amebic dysentery (symptoms include abdominal discomfort, diarrhea, nausea, and possible live abscesses)
Giardia lamblia	Giardiasis (symptoms include abdominal pain, loss of appetite, apathy, headache, and diarrhea alternating with constipation)
Helminths:	
Ascaris lumbricoid	es Ascariasis ("large roundworm" infection)
eggs	Trichuriasis ("whip worm" infection)



Table 5-7 Normal survival periods of pathogenic organism (Vigneswaran and Sundaravadivel 2004)

Time of nothern	Maximum survival time in days (Figures in bracket shows the average/normal survival time)					
Type of pathogen	In feces and sludge	In sewage and freshwater	In soil	On crops		
Viruses:						
Enteroviruses	< 100 (<20)	< 120 (<50)	< 100 (<30)	< 60 (<15)		
Bacteria:						
Fecal coliforms	< 90 (<50)	< 60 (<30)	< 70 (<20)	< 30 (<15)		
Salmonella spp.	< 60 (<30)	< 60 (<30)	< 70 (<20)	< 30 (<15)		
Shigella spp.	< 30 (<10)	< 30 (<10)	-	< 10 (<5)		
Vibrio cholerae	< 30 (<5)	< 30 (<10)	< 20 (<10)	< 5 (<2)		
Protozoa:						
Entamoeba hystolyticalysts	< 30 (<15)	< 30 (<15)	< 20 (<10)	< 10 (<2)		
Helminths:						
Ascaris lumbricoides eggs	many months	many months	many months	< 60 (<30)		

5.4 Metals, Volatiles, Semi-Volatiles, Phenolics, and Polychlorinated Biphenyls (PCBs)

5.4.1 Water Quality Standards

The State of Delaware Surface Water Quality Standards (as amended July 11, 2004) established water quality criteria that protect human health from various carcinogens. Standards are provided for systemic toxicants and human carcinogens for surface waters designated as Public Water Supply Sources (Fish and Water Ingestion Standards) and for surface waters that are not designated as Public Water Supply Sources (Fish Ingestion Only Standards) (DNREC 2004). The no action and ocean outfall alternatives propose a discharge into marine waters, which is not a public water supply and is thus regulated by Fish Ingestion Only Standards.

5.4.2 Priority Pollutant Scan

The City of Rehoboth Beach analyzed the treated effluent from the RBWWTP for priority pollutants as required by DNREC when renewing the NPDES discharge permit for the treatment facility. Three separate 24-hour composite samples of the treated effluent were collected on three different days during June and



July of 2010. The samples were analyzed for 13 metals, 85 volatile and semi-volatile organics, and phenolic compounds. The laboratory results are provided in (Appendix F).

All three effluent samples were analyzed for the parameters listed in Table 5-8 using the indicated EPA laboratory method.

Table 5-8 Analyte Groups and EPA Methods of Priority Pollutant Scan (see (Appendix F))

Analyte Group	EPA Method	Identification/Quantification Equipment
Metals (other than mercury)	200.8	IPC-MS
Mercury	245.1	CVAA
Volatile organic compounds	624	GC/MS
Semi-volatile organic compounds	625	GC/MS
Phenolics	420.4	Semi-automated Colorimetric

As per federal regulations, the samples were not filtered prior to analysis, so detected concentrations include contaminates in the non-dissolved form. The toxic bioavailable form of most pollutants is the dissolved form, which is typically a fraction of the total contaminant concentration. Since the samples were analyzed "whole", the results will generally overstate the ecological and human health risk (Greene 2011).

5.4.3 Polychlorinated Biphenyls (PCB) Scan

Polychlorinated Biphenyls (PCBs) are a class of man-made toxic organic compounds classified as a persistent organic pollutant. PCBs were manufactured and used in electrical equipment such as transformers and capacitors, paints, printing inks, pesticides, hydraulic fluids and lubricants, until federal regulations banned most uses in the late 1970s (DRBC 2006). However, certain exceptions to the ban exist, and PCBs may also be created as a by-product in certain manufacturing processes.

As a persistent organic chemical, PCBs can gather in soils and sediments and concentrate in the tissues of aquatic biota either directly or indirectly through the food chain (DRBC 2006). PCBs have many physiological effects on humans and animals, including dermatologic, reproductive, endocrine, and carcinogenic effects (ATSDR 1990).

There are 209 possible PCB compounds based on the possible configurations of chlorine atoms within the PCB molecule. On September 21, 2011 through September 22, 2011, the City of Rehoboth Beach collected a 24-hour composite sample of the RBWWTP effluent and an equipment rinsate blank on September 21, 2011 prior to sampling. The samples were sent to the lab to be analyzed for PCBs using EPA Method 1668A. Method 1668 is high resolution gas chromatography/mass spectrometry method capable of identifying and quantifying all 209 individual PCBs. The PCB congener results are presented in (Appendix I).



The PCB congener results for the effluent sample, the rinsate blank, and a laboratory method blank were provided to DNREC on October 27, 2011.

5.4.4 Compliance with Water Quality Criteria

5.4.4.1 Metals

Thirteen (13) metals were analyzed in the three effluent samples. In every case the metals were either below detection limits or were present at concentrations substantially below the level of concern as listed in the Surface Water Quality Standards. Table 5-9 lists the metals and the analytical results for all three samples. A result reported as "less than" as indicated by the symbol < means that the parameter could not be identified within the detection limits of the laboratory procedure. Out of the 39 possible detections, there were only 16 detections, yielding an overall detection frequency of 41%.

Table 5-9 Measured Levels of Metals (see (Appendix F))

Lab Result	Lab Result (μg/L)			
June 30	July 6	July 13	Frequency	
< 1	< 1	< 1	0 of 3	
1.0	1.9	< 1	2 of 3	
< 1	< 1	< 1	0 of 3	
< 0.5	< 0.5	< 0.5	0 of 3	
< 1	3.0	2.6	2 of 3	
1.8	7.0	4.6	3 of 3	
< 1	< 1	< 1	0 of 3	
< 0.5	< 0.5	< 0.5	0 of 3	
3.4	6.2	3.8	3 of 3	
1.0	< 1	< 1	1 of 3	
0.75	0.68	< 0.5	2 of 3	
< 1	< 1	< 1	0 of 3	
21.2	46.7	37.4	3 of 3	
	June 30 < 1 1.0 < 1 < 0.5 < 1 1.8 < 1 < 0.5 3.4 1.0 0.75 < 1	June 30 July 6 < 1	June 30 July 6 July 13 < 1	

The concentrations of metals identified by Delaware's Surface Water Quality Standards for the protection of human health and aquatic life are presented in Table 5-10. Even under the more stringent limits imposed on surface waters classified as Public Water Supply Sources (Fish & Water Ingestion), the effluent meets the



surface water quality criteria for human health without dilution. With the exception of copper, all of the detections listed in Table 5-9 are less than the applicable water quality criteria for the protection of aquatic life. However, the criteria listed in Table 5-10 apply to the ambient receiving waters after proper consideration of mixing/dilution and other fate processes. The detected concentration of 7.0 μ g/L copper must undergo at least 1:3 dilution to achieve the required 3.1 μ g/L concentration.

Table 5-10 Water Quality Criteria for Metals (DNREC 2004)

	Criteria for Protection of Human Health (μg/L)				Criteria for Protection of Aquatic Life (µg/L)		
Metal	Systemic Toxicant		Human Carcinogen		Marine	Marine	
	Fish Ingestion	Fish & Water Ingestion	Fish Ingestion	Fish & Water Ingestion	Acute Criterion	Chronic Criterion	
Antimony	1,600	6	-	-	-	-	
Arsenic	-	10	-	-	-	-	
Beryllium	420	4	0.024	0.0034	-		
Cadmium	31	5	-	-	-		
Chromium	-	100	-	-	-	-	
Copper	-	1,300	-	-	4.8	3.1	
Lead	-	15	-	-	210	8.1	
Mercury	-	-	-	-	1.8	0.94	
Nickel	1,700	100	-	-	74.	8.2	
Selenium	4,200	50	-	-	290	71.	
Silver	40,000	170	-	-	1.9		
Thallium	18	2	-	-	-	-	
Zinc	26,000	7,400	-	-	90	81	

5.4.4.2 Volatile Organic Compounds

Thirty-four (34) volatile organic compounds were analyzed in the three effluent samples, with a nominal detection limit of between 1 and 2 μ g/L. Of the 102 possible detections, there was only a single detection yielding an overall detection frequency of less than 1%. The sample results are included in (Appendix F). The detection limits are reported on the sample results as the RDL or Reporting Detection Limit.

The single detection was 1.1 μ g/L of chloroform, a simple trihalomethance with natural and man-made sources (Greene, Review of Rehoboth Beach, DE Sewage Treatment Plant Effluent Data for Toxic



Pollutants 2011). Neither Delaware nor the EPA has aquatic life criteria for chloroform, but the requirements of other states can be utilized to determine the concentration within the effluent that could potentially impact the environment. The California EPA lists a lowest observed effect level (LOEL) of 6,400 μ g/L for chronic effects to saltwater species (California EPA 2011). The concentration of chloroform identified by Delaware's Surface Water Quality Standards imposed on surface waters classified as a public water supply is 340 μ g/L chloroform (DNREC 2004). The single detection of chloroform is orders of magnitude less than both of these criteria, so the effluent meets all human health and aquatic life criteria for volatile organic compounds.

5.4.4.3 Semi-Volatile Organic Compounds

Fifty-four (54) separate semi-volatile organic compounds were analyzed in the three effluent samples, with a nominal detection limit of between 1.4 and 18 μ g/L. Semi-VOAs include many common contaminants such as polyaromatic hydrocarbons (PAHs), phthalates, and phenols. Out of the 162 possible detections, there was only a single detection yielding an overall detection frequency of less than 1%. The sample results are included in (Appendix F). The detection limits are reported on the sample results as the RDL or Reporting Detection Limit.

The single detection was $8.0~\mu g/L$ of bis(2-ethylhexyl) phthalate (BEHP). Neither Delaware nor the EPA has aquatic life criteria for this compound. However, the compound has been determined from aquatic toxicity data to not be toxic to aquatic life below its solubility limit of $29~\mu g/L$ (USEPA 2002) (Schwarzenbach, Gschwend and Imboden 2003). The concentration of BEHP identified by Delaware's Surface Water Quality Standards imposed on surface waters classified as a public water supply is $2.2~\mu g/L$ chloroform (DNREC 2004). The detected concentration of $8.0~\mu g/L$ of BEHP must undergo at least 1:4 dilution to achieve the required concentration limit.

5.4.4.4 Phenolics

Phenolics are a class of compounds that encompass many chemicals, including some compounds identified as estrogenic or endocrine disrupters. Of the three samples taken, only one sample contained a detectable level of phenolics with a reported concentration of 5 µg/L. It is not unusual for phenolics as a class to be detected in effluent, and there is no water quality criterion for phenolics as a class. The only test performed for phenolics was for the overall level; the levels of individual phenolic compounds is not known. Phenolics include many natural and man-made compounds, including the antibacterial agent Triclosan. Triclosan is used in many consumer products, including hand soap, cleaning supplies, deodorants, and mouthwash, though the safety in consumer products is currently under review by the FDA (Greene, Review of Rehoboth Beach, DE Sewage Treatment Plant Effluent Data for Toxic Pollutants 2011).

5.4.4.5 PCB

The RBWWTP effluent was tested for all 209 PCB compounds. A review by the DNREC (Greene 2011a) was performed during November 2011 as detailed below.

The concentration of total PCB in the RBWWTP effluent was 425 pg/L. The PCB concentration in the effluent is well below DNREC's marine chronic aquatic life criterion of 30,000 pg/L (DNREC 2004), even without the benefit of any near-field dilution in the receiving water. The concentration of PCB in the undiluted



effluent does exceed DNREC's applicable human health criterion of 64 pg/L (DNREC 2004), but that criterion does not apply in undiluted effluent. Rather, that criterion applies after proper consideration of dilution in the receiving water. In this case, the criterion would be met after a nominal dilution of 7:1. The near-field dilution of the proposed ocean outfall is expected to be on the order of 50:1 or better. It is therefore concluded that the presence of PCBs in the effluent is not expected to cause an exceedance of the applicable human health water quality criterion after consideration of near-field mixing (Greene, Review of Rehoboth Beach, DE Sewage Treatment Plant Effluent Data for Toxic Pollutants 2011).

In addition to a strict comparison to regulatory criteria, total PCB concentration in the effluent was also compared to total PCB concentrations for similarly-sized municipal WWTPs located within the nearby Delaware River Basin to provide a ranking to other similar discharges. The PCB concentration in the Rehoboth effluent is less than the minimum concentration detected in 95 municipal effluent samples collected from the Delaware River Basin for plants that discharged between 1 and 5 MGD. The minimum among those samples was 578 pg/L compared to the concentration of 425 pg/L in the Rehoboth sample. For further contrast, the mean total PCB concentration in the Delaware River discharges was 6,639 pg/L (Greene 2011a).

The total PCB concentration in the effluent was also compared to available total PCB concentration data for the Atlantic Ocean in the general vicinity of where the ocean outfall is expected to be located to provide an indication of whether the PCB in the effluent will act to increase or decrease existing ambient concentrations. The PCB concentration in the Rehoboth effluent is also less than the PCB concentrations detected in the open waters of the Atlantic Ocean at the mouth of the Delaware Bay. Two samples collected ESE of Dewey Beach, approximately 3.2 miles offshore, had a total PCB concentration of 891 pg/L and 2281 pg/L. One sample was collected in 2006 while the other was collected in 2007. Another sample collected off the coast of Cape May, NY in 2006 had a total PCB concentration of 1612 pg/L. Note that the average among the two samples collected off of Dewey Beach (1,586 pg/L) is quite similar to the single value for Cape May. All of these samples were collected by the Delaware River Basin Commission, and all these samples were analyzed by AXYS Analystical Laboratory using Method 1668A. This lab has the distinction of having developed Method 1668A. To the extent that the PCB concentration in Rehoboth's effluent is less than the ocean water to which it will be discharged, the Rehoboth discharge will slightly improve ambient PCB concentrations in the local ocean water, provided concentrations remain roughly the same or drop at the same rate in the future (Greene 2011a).

Another important calculation was performed with the PCB data. Specifically, PCB congeners were summed by homolog group to better understand the PCB pattern in the samples. PCB homologs are congeners with the same number of chlorine atoms but with different attachment locations on the biphenyl base structure. Since there are 10 possible locations for chlorine atoms to attach to the biphenyl molecule, there are 10 PCB homolog groups. Congeners are homologs with fewer chlorine atoms are lighter, more soluble, and more volatile than congeners and homologs with a greater number of chlorines. This is important because it affects environmental fate. The relative contribution of each homolog group to total PCB was calculated as the ratio of each homolog concentration to total PCB. This provides a type of chemical fingerprint that can be compared among samples and to other known PCB patterns (e.g. pure commercial Aroclor mixtures). The PCB homolog patterns in the sample, the resin blank, and the laboratory blank were all similar with dichlorobiphenyls representing a dominant contribution in all of the samples. These fingerprints are not



typical of any commercial PCB Aroclor mixture. Further, the homolog pattern in the samples is not typical of patterns seen in most other wastewater samples, which are generally dominated by pentachlorobiphenyl. The fact that the PCB mass of the Rehoboth effluent is shifted to the lower molecular weight homologs (which are more volatile), coupled with lower suspended solids concentrations in ocean water (which favors greater partitioning into the dissolved phase), suggests that volatilization to the atmosphere will be an important fate process for the PCB mixture in Rehoboth's effluent. This in turn means that less PCB will be available in local waters near the outfall to be bioaccumulated by fish and other aquatic life (Greene 2011a).

The overall conclusion from this assessment is that the PCBs in Rehoboth's wastewater effluent pose minimal risk to aquatic life and humans. The PCB concentration in Rehoboth's effluent should be considered *de Minimus* by several perspectives (Greene 2011a).

In 2004, Delaware adopted a numerical water quality criterion of 64 pg/L for total PCBs (DRBC 2006). To meet this standard, the detected concentration must undergo 1:12 dilution.

5.5 Pharmaceuticals and Personal Care Products (PPCPs)

5.5.1 Sources of Pharmaceuticals in the Environment

PPCPs in general, are comprised of products used by individuals for personal health or cosmetic reasons or used by agribusiness to enhance the growth or health of livestock. PPCPs comprise a diverse collection of thousands of chemical substances, including prescription and over-the-counter therapeutic drugs, veterinary drugs, fragrances, and cosmetics (USEPA 2010). There are no monitoring requirements in the U.S. and most other countries, but PPCPs have been commonly found in WWTP effluent and many streams and aquifers across the nation. A study conducted by USGS in 30 states in 1999-2000 shows that the concentration of pharmaceuticals, hormones, and other organic wastewater contaminants is generally very low, one part per billion (1 ppb), which is about as much as a tablespoon of water in an Olympic sized swimming pool), and the concentration of PPCPs rarely exceeds drinking-water guidelines, drinking-water health advisories, or aquatic-life criteria (Phillips, et al. 2010) (Kolpin, et al. 2002) (Reynolds 2003).

However, pharmaceutical concentrations could be 10-1000 times higher when a WWTP receives substantial flows (at least 20% of plant flow) from pharmaceutical formulation facilities (Phillips, et al. 2010). Since the service area of RBWWTP is primarily residential with some light commercial consisting of shops and restaurants, it is expected that the concentration of pharmaceutical in its effluent will be very low (1 ppb).

Some of PPCPs are identified as endocrine disruptor compounds. An endocrine disruptor is defined as exogenous agent that interfere with the production, release, metabolism, action, or elimination of natural hormones in the body responsible for the maintenance of homeostasis and the regulation of developmental processes (Kavlock, et al. 1996). These compounds, which include heavy metals (e.g. mercury, lead), a wide variety of anthropogenic organic compounds (e.g. pesticides, plasticizer), steroids and steroid-hormone-mimicking compounds (e.g. synthetic estrogens), may impact survival, growth, and reproduction of numerous animal species (Luckenbach, et al. 2009) (Pait and Nelson 2002).

The presence of estrogen, in natural or synthetic forms, in surface water has been widely reported. A study surveyed coastal marine environments to measure estrogen concentrations shows that the highest values of



estrogen concentration, 30-fold above the background ocean concentration, were found in Rehoboth Bay, Delaware and Key West Harbor, Florida (Atkinson, Atkinson and Tarrant 2003). Both results were from shallow embayments that receive treated WWTP effluent, but WWTP effluent cannot be ruled as the main source of estrogen contamination. Estrogenic chemicals in surface water can be traced back to many sources other than human excretion including industry, agricultural farming, and livestock. Human excretion is only responsible for less than 1% of total estrogen contamination. At least 90% of estrogen contamination is contributed by livestock and other agricultural sources (Wise, O'Brien and Woodruff 2011).

5.5.2 Concentration of Pharmaceuticals in Typical Effluent

Table 5-11 shows the typical concentration of PPCPs found in wastewater influent, the residual concentration after primary and secondary treatments, and the total percent of removal after secondary treatment (Khan and Ongerth 2005).

Table 5-11 Pharmaceuticals residual concentration (Khan and Ongerth 2005).

PPCPs		Primary	Secondary trea	atment	Total
	Influent (µg/L)	treatment Effluent (µg/L)	Aeration Tank Effluent (μg/L)	Clarifier Effluent (µg/L)	removal (%)
Paracetamol/Acetam inophen	76	74	38	35	54%
Metformin (Hydrochloride)	37	36	23	22	41%
Lactulose	1	1	3.00E-01	3.00E-01	70%
Amoxycillin	14	14	6	5	64%
Ranitidine (Hydrochloride)	5	5	4	4	20%
Cephalexin	11	11	4	3	73%
Naproxen	5	5	2	2	60%
Valproate (Sodium)	1	1	6.00E-01	5.00E-01	50%
Aspirin	1.00E-03	1.00E-03	6.00E-04	5.00E-04	50%
Salicylic acid (Ex- Aspirin)	4	4	2	2	50%
Gemfibrozil	8	8	7	4	50%
Allopurinol	3	3	2	2	33%
	Paracetamol/Acetam inophen Metformin (Hydrochloride) Lactulose Amoxycillin Ranitidine (Hydrochloride) Cephalexin Naproxen Valproate (Sodium) Aspirin Salicylic acid (Ex-Aspirin) Gemfibrozil	Paracetamol/Acetam inophen 76 Metformin (Hydrochloride) 37 Lactulose 1 Amoxycillin 14 Ranitidine (Hydrochloride) 5 Cephalexin 11 Naproxen 5 Valproate (Sodium) 1 Aspirin 1.00E-03 Salicylic acid (Ex-Aspirin) 4 Gemfibrozil 8	PPCPsInfluent (μg/L)treatment Effluent (μg/L)Paracetamol/Acetam inophen7674Metformin (Hydrochloride)3736Lactulose11Amoxycillin1414Ranitidine (Hydrochloride)55Cephalexin1111Naproxen55Valproate (Sodium)11Aspirin1.00E-031.00E-03Salicylic acid (Ex-Aspirin)44Gemfibrozil88	PPCPs Influent (μg/L) Freatment Effluent (μg/L) Aeration Tank Effluent (μg/L) Paracetamol/Acetam inophen 76 74 38 Metformin (Hydrochloride) 37 36 23 Lactulose 1 1 3.00E-01 Amoxycillin 14 14 6 Ranitidine (Hydrochloride) 5 5 4 Cephalexin 11 11 4 Naproxen 5 5 2 Valproate (Sodium) 1 1 6.00E-01 Aspirin 1.00E-03 1.00E-03 6.00E-04 Salicylic acid (Ex-Aspirin) 4 4 2 Gemfibrozil 8 8 7	PPCPs Influent (μg/L) treatment Effluent (μg/L) Aeration Tank Effluent (μg/L) Clarifier Effluent (μg/L) Paracetamol/Acetam inophen 76 74 38 35 Metformin (Hydrochloride) 37 36 23 22 Lactulose 1 1 3.00E-01 3.00E-01 Amoxycillin 14 14 6 5 Ranitidine (Hydrochloride) 5 5 4 4 Cephalexin 11 11 4 3 Naproxen 5 5 2 2 Valproate (Sodium) 1 1 6.00E-01 5.00E-01 Aspirin 1.00E-03 1.00E-03 6.00E-04 5.00E-04 Salicylic acid (Ex-Aspirin) 4 4 2 2 Gemfibrozil 8 8 7 4



No. PPCPs Influent (μg/L) treatment (μg/L) Aeration Tank Effluent (μg/L) Clarifier Effluent (μg/L) removal removal removal removal (%) 11b Oxipurinol (ex-Allopurinol) 8 8 5 4 50% 12 Sulphasalazine 9.00E-04 9.00E-04 8.00E-04 7.00E-04 22% 13 Ibuprofen 9.00E-01 9.00E-01 5.00E-01 5.00E-01 44% 14 Chlorothiazide 6 6 5 5 17% 15 Quinine (Sulphate) 1 5.00E-01 4.00E-01 4.00E-01 60% 16 Erythromycin 2.00E-01 2.00E-01 3.00E-01 2.00E-01 0% 17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02		PPCPs	Influent (µg/L)	Effluent	Secondary tre	_ Total	
Sulphasalazine 9.00E-04 9.00E-04 8.00E-04 7.00E-04 22%	No.				Tank Effluent	Effluent	removal
13 Ibuprofen 9.00E-01 9.00E-01 5.00E-01 5.00E-01 44% 14 Chlorothiazide 6 6 5 5 17% 15 Quinine (Sulphate) 1 5.00E-01 4.00E-01 4.00E-01 60% 16 Erythromycin 2.00E-01 2.00E-01 3.00E-01 2.00E-01 0% 17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-02 10% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 1 1 0 <td< td=""><td>11b</td><td>Oxipurinol (ex- Allopurinol)</td><td>8</td><td>8</td><td>5</td><td>4</td><td>50%</td></td<>	11b	Oxipurinol (ex- Allopurinol)	8	8	5	4	50%
14 Chlorothiazide 6 6 5 5 17% 15 Quinine (Sulphate) 1 5.00E-01 4.00E-01 4.00E-01 60% 16 Erythromycin 2.00E-01 2.00E-01 3.00E-01 2.00E-01 0% 17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Dittiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 6.00E	12	Sulphasalazine	9.00E-04	9.00E-04	8.00E-04	7.00E-04	22%
15 Quinine (Sulphate) 1 5.00E-01 4.00E-01 4.00E-01 60% 16 Erythromycin 2.00E-01 2.00E-01 3.00E-01 2.00E-01 0% 17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67%	13	Ibuprofen	9.00E-01	9.00E-01	5.00E-01	5.00E-01	44%
16 Erythromycin 2.00E-01 2.00E-01 3.00E-01 2.00E-01 0% 17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27	14	Chlorothiazide	6	6	5	5	17%
17 Cefaclor 4 4 1 1 75% 18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 <	15	Quinine (Sulphate)	1	5.00E-01	4.00E-01	4.00E-01	60%
18 Carbamazepine 2.00E-01 2.00E-01 1.00E-01 1.00E-01 50% 19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-02 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin	16	Erythromycin	2.00E-01	2.00E-01	3.00E-01	2.00E-01	0%
19 Verapamil (Hydrochloride) 8.00E-01 8.00E-01 8.00E-01 0% 20 Moclobemide 5.00E-02 5.00E-02 4.00E-02 4.00E-02 20% 21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1	17	Cefaclor	4	4	1	1	75%
19	18	Carbamazepine	2.00E-01	2.00E-01	1.00E-01	1.00E-01	50%
21 Phenoxymethylpenic illin 1 1 6.00E-01 5.00E-01 50% 22 Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	19		8.00E-01	8.00E-01	8.00E-01	8.00E-01	0%
Diltiazem (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10%	20	Moclobemide	5.00E-02	5.00E-02	4.00E-02	4.00E-02	20%
22 (Hydrochloride) 1.00E-01 1.00E-01 9.00E-02 9.00E-02 10% 23 Sulphamethoxazole 1 1 9.00E-01 9.00E-01 10% 24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	21		1	1	6.00E-01	5.00E-01	50%
24 Gliclazide 1 1 1 1 0% 25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	22		1.00E-01	1.00E-01	9.00E-02	9.00E-02	10%
25 Methyldopa 3 3 1 1 67% 26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 6.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	23	Sulphamethoxazole	1	1	9.00E-01	9.00E-01	10%
26 Metoprolol (Tartrate) 9.00E-02 9.00E-02 6.00E-02 5.00E-02 44% 27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	24	Gliclazide	1	1	1	1	0%
27 Frusemide 2 2 2 2 0% 28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	25	Methyldopa	3	3	1	1	67%
28 Atenolol 3 2 1 1 67% 29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	26	Metoprolol (Tartrate)	9.00E-02	9.00E-02	6.00E-02	5.00E-02	44%
29 Flucloxacillin 1 1 9.00E-01 9.00E-01 10% 30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	27	Frusemide	2	2	2	2	0%
30 Cimetidine 1 1 9.00E-01 9.00E-01 10% 31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	28	Atenolol	3	2	1	1	67%
31 Ketoprofen 1 1 6.00E-01 5.00E-01 50%	29	Flucloxacillin	1	1	9.00E-01	9.00E-01	10%
	30	Cimetidine	1	1	9.00E-01	9.00E-01	10%
32 Phenytoin 2.00E-04 2.00E-04 2.00E-04 0%	31	Ketoprofen	1	1	6.00E-01	5.00E-01	50%
	32	Phenytoin	2.00E-04	2.00E-04	2.00E-04	2.00E-04	0%



No.	PPCPs	Influent (µg/L)	Primary treatment Effluent (µg/L)	Secondary trea	_ Total	
				Aeration Tank Effluent (μg/L)	Clarifier Effluent (µg/L)	removal (%)
33	Diclofenac	2.00E-01	2.00E-01	2.00E-01	1.00E-01	50%
34	Codeine (Phosphate)	2.00E-01	2.00E-01	1.00E-01	1.00E-01	50%
35	Clavulanic Acid	6.00E-01	6.00E-01	2.00E-01	2.00E-01	67%
36	Roxithromycin	1	1	2	1	0%
37	Irbesartan	2	8.00E-01	2	2.00E-02	99%
38	Sertraline	3.00E-03	3.00E-03	4.00E-03	3.00E-03	0%
39	Dicloxacillin	1	1	7.00E-01	7.00E-01	30%
40	Metronidazole	4.00E-01	4.00E-01	3.00E-01	3.00E-01	25%
41	Captopril	7.00E-01	7.00E-01	2.00E-01	2.00E-01	71%
42	Trimethoprim	6.00E-01	6.00E-01	5.00E-01	4.00E-01	33%
43	Isosorbide Mononitrate	1.00E-04	1.00E-04	8.00E-05	8.00E-05	20%
44	Nizatidine	8.00E-01	8.00E-01	7.00E-01	7.00E-01	13%
45	Tiaprofenic Acid	7.00E-01	7.00E-01	4.00E-01	4.00E-01	43%
46	Dothiepin (Hydrochloride)	1.00E-01	1.00E-01	1.00E-01	8.00E-02	20%
47	Simvastatin	9.00E-01	5.00E-01	9.00E-01	3.00E-02	97%
48	Hydrochlorothiazide	1	1	1	1	0%
49	Sotalol (Hydrochloride)	7.00E-01	7.00E-01	5.00E-01	4.00E-01	43%
50	Doxycycline	3.00E-01	3.00E-01	3.00E-01	3.00E-01	0%
	·	·	· · · · · · · · · · · · · · · · · · ·	·		· · · · · · · · · · · · · · · · · · ·

Additional tertiary treatment (disinfection), such as chlorination, UV, and ozonation could also help to further reduce the residual concentration of PPCPs. Table 5-12 shows the effectiveness of different disinfection processes for PPCP removal (Snyder, Wert, et al. 2007). Currently, disinfection at RBWWTP is achieved by chlorination.



Table 5-12 Efficiencies of PPCPs removal with Tertiary Treatments Processes/Disinfection (Snyder, Wert, et al. 2007)

No.	222	Total removal						
NO.	PPCPs	Chlorination	Chloramination	UV treatments	Ozonation			
1	Caffeine	<20%	<20%	<20%	>80%			
2	Carbamazepine	<20%	<20%	<20%	>95%			
3	Diclofenac	>80%	50-80%	50-80%	>95%			
4	Gemfibrozil	50-80%	<20%	<20%	>95%			
5	Ibuprofen	<20%	<20%	<20%	50-80%			
6	Sulfatmethoxazole	>80%	<20%	50-80%	>95%			
7	TCEP	<20%	<20%	<20%	<20%			
8	Triclosan	>80%	>80%	50-80%	>95%			

Chlorination is a disinfection process using free chlorine. Free chlorine is a powerful oxidant and reacts rapidly with organics and inorganics. However, chlorine is relatively unstable in water and could dissipate to the atmosphere on its own. The chlorination experiments mentioned in Table 5-12 were performed using liquid sodium hypochlorite (NaOCI). Chlorine doses were determined based upon the chlorine demand in order to achieve a residual goal of approximately 0.5 mg/L after 24 hours. After 24 hour contact time, residual chlorine was guenched with 50 mg/L of ascorbic acid (Snyder, Wert, et al. 2007).

Chloramination is the process of disinfecting water using chloramines, a compound containing both chlorine and ammonia, which is much more stable than chlorine. Chloramination experiments mentioned in Table 5-12 were performed by first adding ammonia to raw water followed by sodium hypochlorite (NaOCI). A chlorine:ammonia ratio of 4:1 was used because this is commonly used in drinking water treatment. This sequence of chemical addition was selected over preformed monochloramine solution to closely simulate actual full-scale plant conditions while minimizing exposure to free chlorine. As a result, dichloramine and monochloramine are likely formed, resulting in higher target compound removal. The experiment was conducted in room temperature and ambient pH. The chloramine residual was quenched with ascorbic acid (Snyder, Wert, et al. 2007).

Ultraviolet (UV) light treatment is another alternative for organic pollutant removal from contaminated waters. Ultraviolet light oxidizes organics in water via two mechanisms. First, UV can directly cleave bonds in organic molecules by direct photolysis. Second, UV reacts with water or inorganic constituents in water to form highly reactive intermediates, with the formation of hydroxyl radicals. The UV treatment experiments mentioned in Table 5-12 were performed using two different collimated beam systems from two manufacturers. Both systems used a medium pressure lamp. UV fluences were calculated using the incident irradiance, sample geometry, and water absorption spectrum (Snyder, Wert, et al. 2007).



Ozonation is a strong oxidant and disinfectant. Unlike free chlorine or chloramine, ozone decays rapidly within minutes after its addition to water and results in the formation of fewer halogenated organic disinfection by-products. Ozone reacts with organic contaminants through either the direct reaction with molecular ozone or through the formation of free radicals, including the hydroxyl radical. The ozonation experiments mentioned in Table 5-12 were conducted by injecting a high concentration of dissolved ozone into source water. Dissolved ozone solutions were made by dissolving a high concentration of gaseous ozone into deionized water at 2oC. All dissolved ozone residuals were measured using the indigo method. Ozone doses were determined using an ozone demand/decay curve generated for each water type (Snyder, Wert, et al. 2007).

5.5.3 Pharmaceutical Impacts to Humans

To date, there is no health impact of human exposure to traces of pharmaceuticals found in the surface water ever reported. There is, however, known mammalian effects for certain drug classes that justify the need for further investigation. Various antiepileptic drugs (e.g., phenytoin, valproate, and carbamazepine, the latter of which is frequently identified in sewage effluents) are becoming more recognized as human neuroteratogens, triggering extensive apoptosis in the developing brain (during critical developmental "exposure windows"), and consequently leading to general neurodegeneration (Daughton 2001). A joint study of researchers in the Netherland, Germany, and Finland founds the relationship between the use of antiepileptic drugs (e.g., phenytoin, valproate, and carbamazepine, the latter of which is frequently identified in sewage effluents) during pregnancy and the risk of major congenital malformations, which is a physical defect present in a baby at birth (Samrén, et al. 1997). Offspring of mothers using antiepileptic drugs during pregnancy were at a significantly increased risk of major congenital malformations, especially neural tube defects. The risk of major congenital malformations also increased when the mothers using the combination of antiepileptic drugs (phenobarbital and ethosuximide, or phenytoin, phenobarbital, carbamazepine, and valproate). However, it is important to note that the risk of major congenital malformation reported in the study (Samrén, et al. 1997) was observed when the mothers used a relatively high doses of antiepileptic drugs (1000 mg per day), while the typical concentration of antiepileptic drugs found in surface water is only 6.2 nanogram per liter or 3.5 million times lower than the observed dose.

The list of other possible disorders of the human reproductive system involving endocrine disruptive compounds according to Endocrine Society Scientific Statement (Diamanti-Kandarakis, et al. 2009) is shown in Table 5-13.



Table 5-13 Disorder of the human reproductive system possibly involving endocrine disruptor compounds in their pathogenesis: A sexually dimorphic life cycle perspective (Diamanti-Kandarakis, et al. 2009)

	Fetal/neonatal	Prepubertal	Pubertal	Adult
Processes	Intraurine growth	Adrenarche	Gonadarche	Spermatogenesis
	Sexual			Ovulation
	differentiation			Hormonal control of prostate, breast, uterus, and location
Male disorders	Intrauterine growth	Preamature	Small testes and	Oligospermia*
	retardation	pubarce	high FSH (Follicle- stimulating	Testicular cancer*
	Cryptorchidism* Hypospadias*		hormone)	Prostate hyperplasia
			Early puberty	
			Delayed puberty	
Female disorders	Intrauterine growth retardation	Premature thelarce	Secondary central precocious puberty	Vaginal adenocarcinoma
		Peripheral precocious puberty	Polycystic ovarian	Disorders of ovulation
			syndrome	Benign breast disease
		Preamature	Delayed ovulatory cycles	Breast cancer
		pubarce	2, 3.00	Uterine fibroids
				Disturbed lactation

^{*} Cryptorchidism, hypospadias, oligospermia, and testicular cancer are four components of the "testicular dysgenesis syndrome" as a common entity.

A critical concern is the potential lag between exposure to endocrine disruptor compounds and the manifestation of a clinical disorder. In humans, this period may be years or decades. In the case of reproduction, infertility cannot be assessed until the exposed individual has attained a certain age, again resulting in a lag between early exposure and manifestation of a dysfunction (Diamanti-Kandarakis, et al. 2009). Thus, an extensive research effort is needed to explain the real impact of endocrine disruptor compounds to human exposure.

To truly understand the real risk of PPCPs exposure to human, it is important to understand how human body reacts to different concentration of PPCPs. For most chemicals, the rate of change in human response will vary by different doses. The response may change slowly at low doses but rapidly increase at high doses. For compounds other than cancer-causing agents (carcinogens), there is assumed to be a threshold dose below which no effects occur, similar to a drug for which a dose that is too small has no beneficial



effect. For carcinogens, it is assumed that any exposure can increase the potential for developing cancer over a lifetime (Snyder, Vanderford, et al. 2008).

The human exposure to PPCPs through direct contact to treated wastewater effluent is expected to be minimal. However, humans could be more significantly exposed to PPCPs through surface water, ground water, or consumption of drinking water. To put the risk of human exposure to PPCPs into perspective, the concentration of PPCPs found in groundwater, surface water and drinking water should be compared to their associated therapeutic dose, which is the minimum intake to produce a clinical effect. A study reported that, in most circumstances, human exposure rates to a pharmaceutical or combination of pharmaceuticals are at least 100 fold lower than those required to produce minimal therapeutic effects (Kostich and Lazorchak 2008). Since most pharmaceuticals do not cause inherent toxicity except at dosages well above minimum therapeutic dosages, the predicted exposure is not expected to be toxic to human.

Table 5-14 shows the comparison between groundwater or surface water, drinking water, and Drinking Water Equivalent Level (DWEL) therapeutic dose (Snyder, Wert, et al. 2007) (Snyder, Vanderford, et al. 2008). DWEL therapeutic dose is the minimum amount of concentration of PPCP compound to produce a clinical effect assuming drinking water as the only source of PPCP intake. The results show the small risk of PPCP exposure. For example, the minimum therapeutic dose of Carbamazepine (anti epilepsy medicine) is 10 miligram per day for a child, age less than 6 years and assumed bodyweight of 22 pounds. To get this therapeutic dose solely from drinking surface water (with a concentration of 6.2 ng/L Carbamazepine), the child would need to drink over 420,000 gallons of the contaminated water a day.

Table 5-14 PPCP's concentrations in raw drinking water, drinking water, and their Equivalent Level (DWEL) therapeutic dose (Snyder, Wert, et al. 2007) (Snyder, Vanderford, et al. 2008)

	Groundwater or surface water		Drinking water				
PPCPs	# of detected samples (out of 20 samples)	Average concen- tration (ng/L) ¹	# of detected samples (out of 20 samples)	Average concen- tration (ng/L)	Minimum thera-peutic dose (mg/d)	Age recom- mender dose based on	
Carba-mazepine	18	6.2	11	2.8	10	Child (age less than 6 years)	
Sulfa- methoxazole	17	14.0	1	20	400	Child (age greater than 2 months)	
Ibuprofen	16	6.1	13	7.9	50	Child (age 6-12 months)	
Gemfibrozil	13	5.2	5	3.9	1,200	Adult	
Naproxen	10	5.7	1	8	125	Adult	



	Groundwater or surface water		Drinking water			
PPCPs	# of detected samples (out of 20 samples)	Average concen- tration (ng/L) ¹	# of detected samples (out of 20 samples)	Average concen- tration (ng/L)	Minimum thera-peutic dose (mg/d)	Age recom- mender dose based on
Aceta- menophen	7	2.7	Not detected	Not detected	160	child (age 2-3 years)
Trimethoprim	3	1.8	1	1.3	80	"child" or "pediatric"

Note:

Another study measured the acceptable daily intakes of pharmaceuticals and used it to estimate the predicted no effect concentrations from two main sources of potential human exposure: drinking water and fish ingestion (Schwab, et al. 2005). The results were then compared to measured environmental concentrations from published literature and maximum predicted environmental concentration (calculated under conservative assumptions of low river flow and no depletion, e.g. no metabolism, no removal during wastewater or drinking water treatment). For all 26 pharmaceutical compounds tested, the results showed no appreciable human health risk exists from the presence of trace concentrations of pharmaceutical in surface water and drinking water (Schwab, et al. 2005).

5.5.4 Impact of PPCP exposure to Non-human species

While the low levels of pharmaceuticals are unable to induce acute effects in humans, its impact on other non-human organisms is evident (Reynolds, Pharmaceuticals in Drinking Water Supplies 2003). Due to a much smaller body mass, small biota are more sensitive to low concentrations of pharmaceuticals.

5.5.4.1 PPCPs impacts on marine phytoplankton

DeLorenzo and Fleming (2008) analyzed the individual and mixture toxicity of six PPCPs (including Simvastatin, clofibric acid, diclofenac, carbamazepine, fluoxetine, and triclosan) on the marine phytoplankton species *Dunaliella tertiolecta*. Among them, only triclosan yielded toxicity at typical environmental concentrations. Detrimental effects of pharmaceuticals on phytoplankton populations could ultimately impact nutrient cycling and food availability to biota at a higher level of the food chain. However, more detailed research is required to explain how and to what degree accumulation of PPCPs will impact biota at higher levels of the food chain.

^{1.} $ng/L = nanogram/Liter = 10^{-9} g/L$



5.5.4.2 Endocrine disruptor impacts on freshwater fish

The presence of estrogen in water has been linked to the phenomenon of male fish feminization found in English rivers. Extensive laboratory data sets confirm that estrogens are capable of eliciting the effects observed in wild fish at concentrations that have been measured in effluents and in the environment (Gross-Sorokin, Roast and Brighty 2006). Desforges, et al. (2010) compiled data of male teleost fish from 43 rivers on 3 continents and found 28% correlation between the patterns of male production of serum egg protein (vitellogenin) with upstream human population. The insignificant level of population and flow rate ratio with vitellogenin may be explained by the low solubility of estrogenic compounds, resulting in localized contamination near WWTP outfalls, rather than dilution by river water. The same phenomenon of fish feminization was also found at St. Paul, MN. Folmar, et al. (1996) found that the male common carp (Cyprinus caprio) collected from an effluent channel below the St. Paul metropolitan sewage treatment plant had a significantly elevated vitellogenin concentration. However, vitellogenin induction was not observed at any other sampling location, even in fish collected between 3-17 miles downstream of the effluent channel. The dilution of wastewater effluent helps to reduce the exposure to endocrine disruptor compounds. The study also found a significant decrease of serum testosterone levels among male common carp collected in all sampling area, including fish collected from the minimally polluted St. Croix River which is classified as a National Wild and Scenic River.

5.5.4.3 Endocrine disruptor impacts on estuarine/marine fish

Johnson, et al. (1988) conducted a study to assess the impact of polluted ocean water on female English sole (*Parophrys vetulus*) from Puget Sound, WA. Samples from three highly contaminated areas (Duwamish Waterway, Eagle Harbor, and Sinclair Inlet) are compared to minimally contaminated area (Port Susan). The main endocrine disruptor compounds found in Puget Sound are PAHs and PCBs. The results showed that the natural estrogen (E₂) levels of samples from highly contaminated areas are significantly lower than samples from minimally contaminated areas. It was concluded that exposure to endocrine disruptor compounds may interfere with ovarian development in female English sole.

In 1997-2001, as part of the Washington State's Puget Sound Assessment and Monitoring Program, samples of male English sole were collected from 16 sites for evidence of xenoestrogen exposure, using vitellogenin production in males as an indicator. The results of this study indicate that exposure of flatfish to xenoestrogens in Puget Sound is widespread, and provides one of the few reported cases of altered reproductive cycling in association with xenoestrogen exposure in a wild marine fish. Vitellogenin induction in male English sole was observed at three-fourths of the 16 sites sampled in the study. The exposures were greatest near urban centers with high inputs of stormwater and industrial discharges, and at sites near combined sewer overflows and/or sewage treatment plant discharges (Johnson, et al. 2008).

Another study by Collier, et al. (1998) found precocious sexual maturation in 40-50% of juvenile female English sole in Hylebos Waterway in central Puget Sound, WA. In the same location, approximately 20-30% adult female English sole had inhibited gonadal development. These effects were associated with exposure to chlorinated hydrocarbon and PAHs. The reproductive injuries observed in this study presumably would reduce the number of eggs and larvae contributed by these fish to overall fish population of English sole.



5.5.4.4 Excess antibiotics impacts on surface and groundwater

Another important PPCPs contaminant is excess antibiotics. Several stream surveys reported the significant prevalence of native bacteria that display resistance to a wide array of antibiotics including samples from the Mississippi, the Ohio, and the Colorado that tested resistant to ampicillin, a synthetic penicillin (Rallof 1999). In another study Nagulapally (2007) found antibiotic resistant bacteria in municipal wastewater treatment plant influent, secondary clarifier effluent and disinfected effluent. These bacteria (fecal coliforms, Escherichia coli and enterococci) tested resistant to several antibiotics including ciprofloxacin and sulfamethoxazole/trimethoprim. Underwood, et al. (2011) reported a noticeable slower enrichment of groundwater bacterial under antimicrobial agent exposure. Even subtherapeutic concentrations of sulfamethoxazole could alter the composition of the enriched nitrate-reducing microcosms and inhibited nitrate reduction capabilities. Antimicrobial agents were found to affect the growth of natural freshwater algal upstream and downstream of the Olathe, Kansas WWTP (Wilson, et al. 2003).

5.6 Salinity

As discussed in Chapter 3 of Appendix J and shown in the Vertical Salinity Profiles shown in Appendix J, ocean salinity near the ocean floor, where the outfall is proposed to be built, typically varies between 30 and 31 practical salinity units (psu). Closer to the surface, salinity was significantly lower, dropping to as low as 20 psu at multiple sample locations multiple times throughout the year.

As discussed in Section 6.6, near field dilution, as modeled by CORMIX, will provide significant mixing of the effluent with ambient seawater in the immediate vicinity of the outfall. Assuming an ocean salinity of 30 psu at the ocean outfall and an effluent salinity of 0 psu, ocean salinity in the vicinity of the proposed outfall will remain above the observed salinity at the ocean surface (20 psu) if at least 1:3 dilution is achieved. As can be seen in Table 6.9 of this report, the dilution achieved during the most common conditions is 1:930 (Case 3) with a minimum worst case dilution of 1:82. Thus, in all cases, mixing at the outfall will not significantly lower seawater salinity.